



THOMAS HARTMAN FOUNDATION FOR PARKINSON'S RESEARCH, INC.

NEWSLETTER

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ACTRESS SUSAN LUCCI SHOOTS PSA, WORKS WITH HARTMAN FOUNDATION TO SPREAD AWARENESS OF PARKINSON'S DISEASE AND THE CURE FOR SURE DINNER



Garden City, NY (Tuesday, March 23, 2010) - Emmy Award winning actress Susan Lucci, together with the Thomas Hartman Foundation for Parkinson's Disease, is helping to increase awareness and understanding of Parkinson's through a series of Public Service Announcements featuring Ms. Lucci. The PSA's will air on Cablevision, WLNY, and Radio. They are designed to help raise funds for the Hartman Foundation, as well as to let the public know about the "Cure For Sure" dinner which will take place at the Crest Hollow Country Club on June 22 at 6:00 P.M. Ms. Lucci has been a familiar face at the Hartman Foundation since it's inception in 2004 and will be recognized for her ongoing support when she receives the "Philanthropic Honoree" Award at the dinner. Funds raised will support ongoing research that holds the promise of finding a cure for Parkinson's disease.

7th Annual Cure for Sure Dinner

Tuesday, June 22, 2010

Crest Hollow Country Club

For more information, please contact us at 631-277-9655

THOMAS HARTMAN FOUNDATION SCIENTIFIC GRANT AWARD RECIPIENTS

The unique Thomas Hartman Foundation Scientific Research model focuses on key scientific leaders who promote collaboration and foster natural synergies with unparalleled results.

WORLD RENOWNED SCIENTISTS PRESENT THEIR LATEST RESEARCH THAT WILL HELP LEAD TO A CURE FOR PARKINSON'S DISEASE



David Eidelberg, M.D.

The Feinstein Institute for Medical Research

Project Title: Metabolic Networks and the Cognitive Treatment Response in Parkinson's disease.

This study defines the functional circuitry that mediates the cognitive response to Dopaminergic therapy in Parkinson's disease. Very little is understood about the nature of cognitive decline or response therapy in Parkinson's disease. Given the importance of cognitive decline in PD, and both the positive and the negative impacts of pharmacotherapies on this problem, having an objective measure of cognitive dysfunction in the living PD brain and demonstrating that this measure correlates well with responses to therapy would be highly significant. This study is done in humans by Dr. Eidelberg and his team who are the leading group in the world in imaging biomarkers. His approach is safe and sensitive. Biomarkers of cognition are desperately needed. This is cutting edge research and will lead the field to identify new ways to diagnose and treat cognition impairment in PD. In April 2010, Dr. Eidelberg received the movement disorders Research Award in recognition of his outstanding achievements.



D. James Surmeier, Ph.D.
Northwestern University

Project Title: Determinants of Neuronal Vulnerability in Parkinson's disease.

This study will extend his initial findings that the firing of autonomous pacemaker neurons in the substantia nigra with a specific type of calcium channel have sustained elevations of calcium which lead to toxicity and cell death, and that existing anti-hypertensive drugs can block this and thereby reduce dopamine neuronal death. This study will be extended to locus coeruleus neurons which Hartman Foundation grantee Dr. Cliff Saper has found is a brainstem area also degenerated in PD and may influence many non-motor symptoms of PD. This work could explain the long-puzzling predilection of PD for certain cell types in the brain. It may also be the basis for developing new drugs that could help prevent cell loss in PD. In February 2010, Dr. Surmeier was elected as a fellow of the American Association for the Advancement of Science for his achievements and contributions to Science and Technology.

Serge Przedborski, M.D., Ph.D.
Columbia University

Project Title: Parkin and PINK1 —Involvement in Normal Mitochondrial Turnover By Mitophagy.

Parkin mutations lead to a recessive form of PD. How Parkin functions is critical to understanding therapeutics for PD. Parkin and PINK1 are two proteins which have been implicated in the pathogenesis of PD. This study will examine the role of these proteins and their interaction in the long term regulation of degradation of mitochondria. Mitochondria are the cell's main energy source. The role of the mitochondria in cell death is a major area of current neural cell biology. The observations that PINK1 and Parkin mutants both are involved in the degradation of damaged mitochondria means that discovering the methods by which they work has strong potential for helping us to better understand these inherited forms of PD, and may be useful in helping to avoid cell death if we can learn to control the process. The outlined studies use state of the art biochemistry and cell biology to establish the role of Parkin in mitophagy.



Clifford B. Saper, M.D., Ph.D.
Beth Israel Deaconess Medical Center

Project Title: Neuropathy of REM Behavior Disorder

Studies symptoms of Parkinson's disease called non-motor symptoms in which the underlying pathology may involve distinct cell groups of brainstem and spinal cord. This study investigates the presence of alpha-synuclein and/or loss of neurons which may be associated with the onset of REM Behavior Disorder symptoms. Studies show that 80% of patients with this disorder will develop Parkinson's disease. REM behavior disorder (RBD) causes patients to demonstratively act out their dreams by impairing the normal inhibitory effects of sleep on the body's ability to move. If you have a marker for early Parkinson's, you could potentially start a treatment that slows down its progression at an earlier stage.



Rajiv R. Ratan, M.D., Ph.D.
Winifred Masterson Burke Medical Research Institute
Project Title: Investigation of the Efficacy and Mechanism of FDA Approved Activators of Hypoxic Adaptation in the Metabolic Consequences and Treatment of Parkinson's disease.

The goal of his study is to understand how iron chelators prevent oxidative neuronal death. Therapeutic interventions will be developed with already approved FDA treatments to overcome toxicity. As the brain is confronted with stress, (i.e.: free radicals, lack of energy in dopaminergic cells) body mechanisms compensate for that stress. Disease is failure of the body to compensate. The goal of this study is to utilize already approved safe drugs to push the body to do naturally what it would want to do and thereby halt the progression of Parkinson's disease.





David C. Chan, M.D., Ph.D.
California Institute of Technology
Project Title: Mitochondrial Dynamics in Parkinson's disease.

This study is to understand molecular pathways involved in Parkinson's disease and the development of systems to access Mitochondrial Dynamics in the Substantia Nigra (midbrain). Recently, both PINK1 and Parkin were shown to be important for mitophagy (cell degradation and death). This finding will contribute greatly to the understanding of the pathogenesis of Parkinson's disease. Dr. Chan has confirmed that the degradation of these proteins occurs in the Parkin-dependant process, and is investigating how these changes might contribute to mediating mitophagy.

Michael Kaplitt, M.D., Ph.D.
Weill Cornell Medical College of Cornell University
Project Title: Mitochondrial Unfolded Protein Response and Parkinson's disease.

Studies abnormal function of mitochondria and their role in cell death. The goal of his work is to study how the mitochondria, the energy packets inside cells, are involved in Parkinson's disease. Dr. Kaplitt has stated that the following findings would never have happened with the Thomas Hartman Collaborative Model in place. Dr. Michael Kaplitt and Dr. Serge Przedborski discovered that their work reproduced each other and was able to be extended during the funding period which allowed them to go from theory to practical application; their collaborative efforts produced synergistic results that influenced each other. A paper is being published linking the significance of Clp Protein and PINK1.



Andrew Feigin, M.D.
Feinstein Institute for Medical Research of North Shore
Project Title: The Molecular Basis for Cognitive Impairment for Parkinson's disease: A PET Study.



Parkinson's disease (PD) is characterized by progressive motor disability. However, cognitive problems also occur and can have a major impact on patients' quality of life. In fact, even PD patients who do not meet clinical criteria for dementia may demonstrate cognitive deficits on careful neuropsychological testing. The precise cause of these cognitive deficits is not currently known. Our study is utilizing novel imaging methods (multi-tracer positron emission tomography (PET) to investigate the underlying cause of cognitive impairment in non-demented PD patients. We hypothesize that abnormal protein deposits in critical brain regions are responsible for this manifestation of PD. Specifically, we are utilizing (18F)-FDDNP, a PET tracer that binds to aggregates of alpha-synuclein as occur in Lewy bodies (the pathological hallmark of PD). Because this tracer also binds to other protein aggregates such as beta-amyloid (a hallmark of Alzheimer's disease), we are also scanning patients with (11C)-PIB, which is specific for this type of aggregate. We will correlate these PET measures with cognitive performance and with metabolic activity in key brain areas. Our preliminary results suggests that the presence of beta-amyloid in specific cortical regions predicts responsiveness to medications for cognitive dysfunction. This ongoing study will allow us to determine whether observed abnormalities in cognitive performance and brain function in PD are attributable to localized protein aggregates, and whether these changes can reliably predict responses to medications. Our data will be the basis for further studies evaluating new therapies for cognitive symptoms of PD.